Arteries and Veins

Introduction

Blood vessels carry blood. Arteries carry blood away from the heart in high-pressure systems that can tolerate the systolic ejection pressure of the ventricles. Veins carry blood back to the heart, post-capillary, in low-pressure systems. (Capillaries do more than just carry blood, so they get their own lesson—Hemodynamics #2: *Capillaries*.) Arteries and veins are both vessels. All vessels other than capillaries have the same general arrangement in three layers. What and how much are in those layers varies based on what the vessel does. What the vessel does is a product of its proximity to the heart.

In this lesson, we discuss the structure and function of arteries and veins and touch on the physiology of blood flow. You should connect the histology to the purpose of each vessel—connect the physiology of pressure and flow to the microscopic features of the vessel. Figure 1.1 is meant as a summary. Do not let it overwhelm you here at the start. But check back at the end of the lesson and see whether it makes sense.

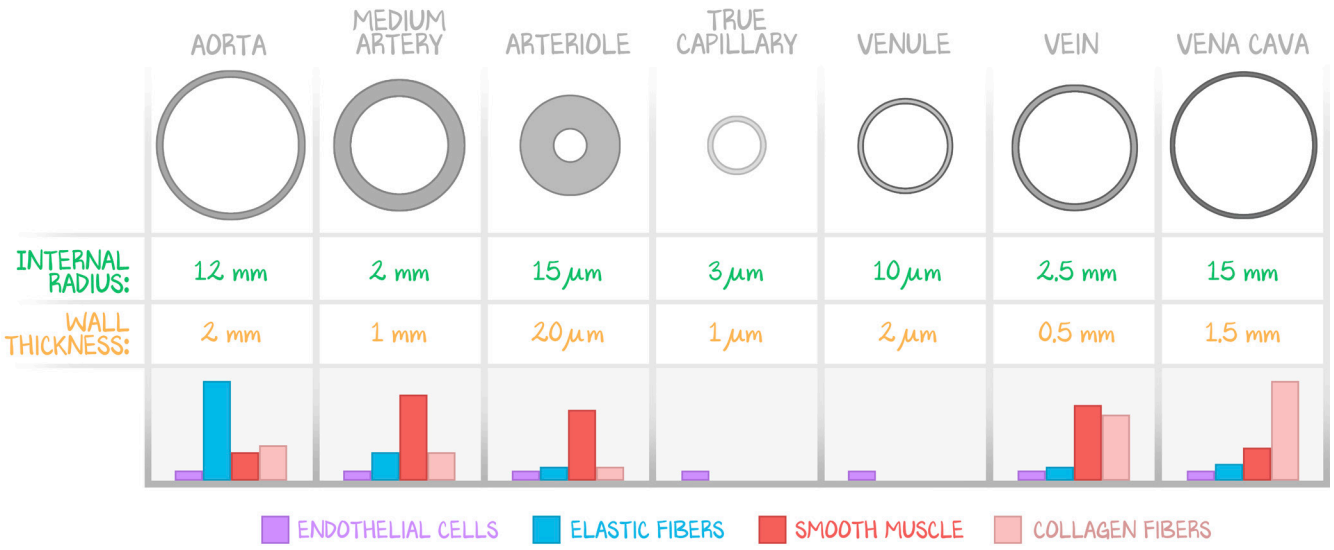

Figure 1.1: Summary of Arteries and Veins
What a vessel is made of depends on the vessel's size, its proximity to the heart, and whether it carries pressurized (arteries) or nonpressurized blood (veins). All vessels have an endothelium. The vessels that need to distend and recoil have an endothelium full of elastin. Those that need to be sturdy when filled are full of collagen. Some vessels have smooth muscles to control their diameter. This chart shows the approximate size (internal radius), how much the vessel can change (wall thickness), and a cross-section of the vessel, then graphs the relative amounts of its likely constituents.

Layers of Vessels

All blood vessels (excluding capillaries) have three layers, called tunicae. Tunica is just a fancy word for layer. Tunicae are separated from one another by an elastic lamina. Blood travels through the center of a blood vessel, called the lumen. *Inside means toward the lumen; outside means away from the lumen.*

The innermost layer is the **tunica intima** (the layer "intimately" close to the lumen) of the blood vessel. The tunica intima should only ever be the **endothelium** and its basement membrane. The endothelium is the epithelium of the blood vessel. It is a single-cell-layer thick, made of endothelial cells—simple squamous epithelium. Everything that carries blood, including capillaries and the chambers of the heart, has an endothelium. No matter how big a vessel gets, it still has only a one-cell-thick endothelium. Because this layer should never be different, regardless of the blood vessel, as we begin to compare the different vessels, we will instead consider the ratio of the internal radius (the radius of the lumen) to the wall thickness.

The **tunica media** (the "middle" layer, between the other two) is the most variable layer and changes based on the vessel's function. The tunica media is full of myofibroblasts. These myofibroblasts can act as **vascular smooth muscle cells** (VSMC), controlling the diameter of the vessel lumen, or as fibroblasts, building, degrading, and recycling the elements of the tunica media's extracellular matrix. The other key components of this layer are fibers of **elastin** and **collagen** (in relative amounts). The tunica media makes both the **internal elastic lamina** (separating it from the tunica intima) and the **external elastic lamina** (separating the tunica media from the tunica adventitia).

Finally, the **tunica adventitia** is the outermost layer of the blood vessel. In very large vessels, such as the aorta, the tunica adventitia carries the vasa vasorum, small arteriole-sized blood vessels that irrigate the tunica media of a blood vessel. In any vessel with a tunica adventitia, this is also where nerves and mesenchymal stromal cells (stem cells that can become connective tissue cells like myofibroblasts and endothelial cells) are.

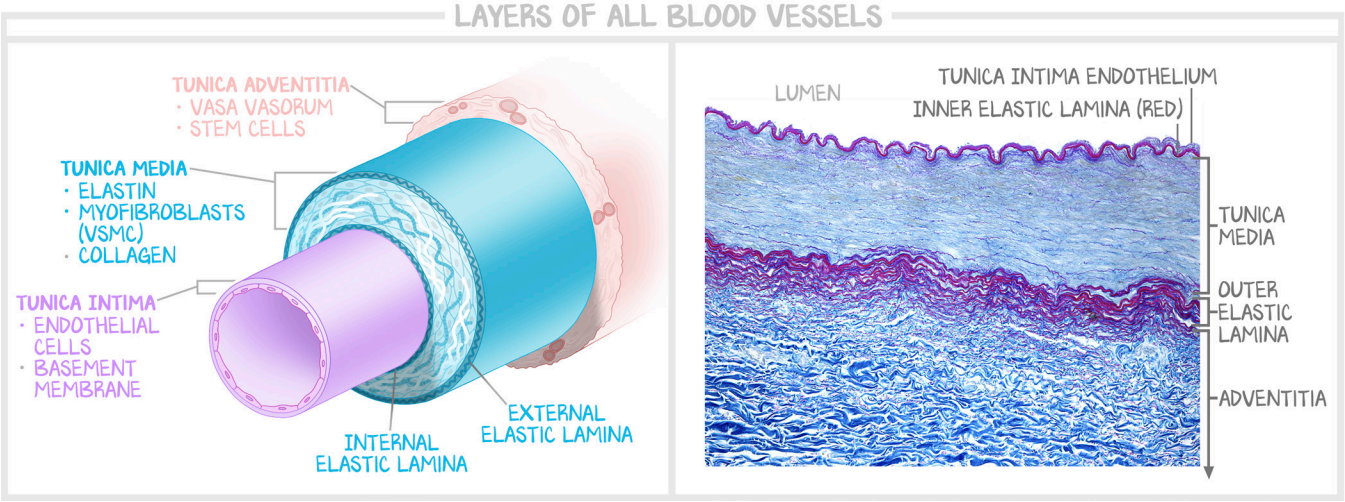

Figure 1.2: The Layers of All Blood Vessels
(a) How we color code the layers of blood vessels. Notice the depiction of the internal elastic lamina and external elastic lamina—they are blue because they are both part of and made by the tunica media. (b) Artery stained for elastin with conspicuous internal and external elastic laminae. Toward the lumen of the inner elastic lamina is the tunica intima, which is normally only the endothelium (as in this image). Between the two elastic laminae is the tunica media. Around the external elastic lamina is the tunica adventitia.

Arteries and Veins at High Level

To do this right, we need to jump ahead a little, to elastin and elastic recoil, and collagen and compliance. **Elastin** enables arteries' **elastic recoil**, and it's the recoil part that is most important. Elastin enables blood vessels distend and then snap back to their original shape. Collagen is stiff and enables blood vessels to resist distension. **Collagen** in arteries prevents them from blowing apart with each contraction and is the chief component of veins. The ability to resist distension is called **compliance**. Arteries resist only a little, so they are said to have high compliance. Veins resist a lot, so they have low compliance.

Arteries carry blood away from the heart to tissues. Being an artery has nothing to do with carrying oxygenated or deoxygenated blood. The pulmonary arteries carry deoxygenated blood from the right ventricle to the lungs; the aorta carries oxygenated blood from the left ventricle to systemic circulation. Arteries are **high-pressure** vessels (which enables **high-velocity flow**) that distend with ventricular contraction and recoil with ventricular relaxation. Said again, arteries have both **high compliance** (they distend) and **high elastic recoil** (they snap back). When the left ventricle contracts (systole),

it pushes blood into the arterial tree; the blood moves forward, down the arterial tree toward the tissues. When the ventricle is not pushing blood down the arterial tree (diastole), blood continues to move forward, down the arterial tree toward the tissues. **Arterial blood is always moving.** This is achieved through arterial **elastic recoil**. Arteries are always filled with blood, and blood is always moving. When the ventricle contracts, it not only pushes blood down the arterial tree but also distends large arteries, like the aorta. They are distended with blood by the force of contraction. When the ventricle stops contracting, the energy stored in the distended large arteries is transferred back to the blood. As the artery recoils back to its pre-distended form, it pushes the blood (which distended it) back into the arterial tree. A valve (the aortic valve) prevents the blood from going back into the left ventricle. And so the blood goes down the arterial tree.

Veins carry blood away from the tissues to the heart. Veins are not pulsatile; they do not distend and recoil. The driving force moving blood forward in the venous system is gravity above the heart and the contraction of skeletal muscle below the heart. Veins are said to be *capacitance vessels* because they have the capacity to fill. Careful with the words—filled and distend. Arteries are always full, and then distend and recoil. Veins are never completely filled with blood except in a pathologic state. They have the capacity to fill, starting as floppy tubes. Once they fill, their **inelastic tunica media**, made inelastic by abundant **collagen**, makes them incapable of filling further. There is no ventricular force or elastic recoil like in the arteries. In fact, skeletal muscle contraction is required to drive the blood from the feet to the heart. That makes veins low-pressure and low-velocity vessels.

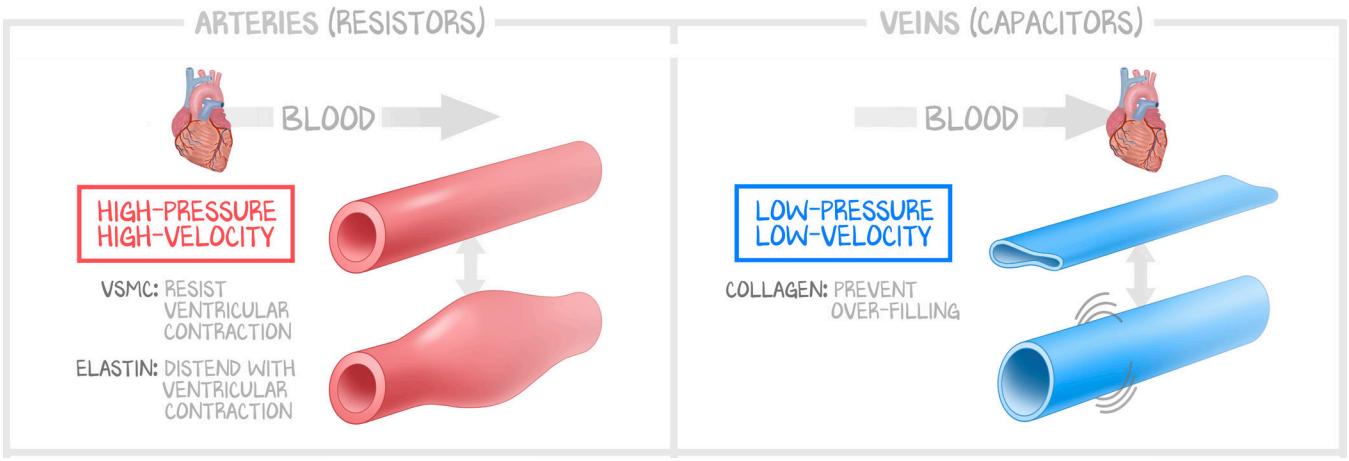

Figure 1.3: Arteries and Vein at a High Level
Arteries use collagen and vascular smooth muscle cells to resist ventricular distention (to keep from blowing apart) and use abundant elastin to snap back to normal. The high-pressure system drives high-velocity blood flow. Arteries are always filled with blood, and blood is always moving. Veins are a low-pressure and low-velocity system. There is no ventricular contraction to distend them, thus there is no need for elastic recoil. Instead, large amounts of collagen resist overfilling. Veins are never completely filled unless in a pathologic state. They have the capacity to be filled (they are floppy) when normal, but when they fill, they don't distend like arteries do. Arteries are pulsatile, distending and recoiling with each heartbeat. Veins are not pulsatile and do not distend and recoil. We've included resistor and capacitor because you will see this elsewhere, but as the next paragraph states, it isn't a great analogy.

Because veins are capacitance vessels, they are called capacitors. In physics, capacitance and resistance have something to do with one another. The natural conclusion, then, is that because veins are capacitors, arteries must be resistors. Wrong. Arteries distend and recoil to sustain the perfusion pressure. Arterioles (more on this in the next section) control the resistance by contracting and dilating their lumen. Veins are floppy and can be filled with blood. The only resistor is the arteriole, which we explore in the next section.

Mean Arterial Pressure

Pressure pushes blood forward. The pressure that moves blood down the arteriole tree toward the tissues is controlled by the driving force of ventricular contraction (systole) and the recoil of the elastic arteries (diastole). When you feel your own radial pulse, you know that it is . . . pulsatile, which is why we call it a pulse. Having a ventricle contraction in systole and an aortic elastic recoil in diastole minimizes the range of its pulsatility. With just a ventricular contraction in systole, there would be a large driving force for a brief moment, then no driving force at all until the next contraction. Blood is continuously flowing during both systole and diastole because there is a force driving it through the arterial tree all the time. There is more pressure during systole than diastole, but there is always a continuous pressure—the **mean arterial pressure**.

Since the heart spends only a third of its time in systole and two-thirds in diastole, the MAP is not just the average of systole and diastole. 1/3 SYS + 2/3 DIA = MAP. This is why a normal blood pressure of around 120/80 has a normal MAP of around 90. But that's only because the heart rate is normal. If the heart rate increases, the arteries feel systole more often than at normal heart rates. When the pulse is tachycardic, the MAP is closer to the average of the two. We're going to talk about MAP and its regulation in Lesson 3. We've introduced it here so that we don't need physics to talk about arterioles and their role in managing blood pressure, flow, and resistance. Instead of saying blood pressure (which has a systole and diastole), we're going to use MAP (which is one number).

A normal MAP is 90. A capillary normally needs a perfusion pressure of around 10. The MAP of 90 is to ensure that there is sufficient perfusion pressure to deliver blood to all tissues. Arterioles are microscopic and give rise to multiple capillaries. The arteries need to deliver enough perfusion pressure to the arterioles to meet the needs of the tissues that their capillaries serve. The arterioles then take that really high pressure (90) and drop it down to what their capillaries need (10). That pressure will be spent across the length of the capillary. This gives us the high-pressure, high-velocity arteries (MAP 90) and the low-pressure, low-velocity veins (venous pressure 0). This is also why we say only arterioles are resistance vessels.

The arterioles' job is to tank the incoming perfusion pressure so that the capillaries don't explode. Arterioles do that by decreasing their lumen's radius, increasing resistance, therefore limiting flow.

Physiology of Arterioles, Pressure, Flow, and Resistance . . . without Physics

The flow of a fluid through a tube is determined by the driving force pushing it along that tube and the resistance of that tube. You already know this stuff, intuitively. Think of a bathroom sink. If you turn the faucet on, you create a driving force. If the drain is open, the water goes down the drain and doesn't fill the sink. If you plug the drain, the water doesn't go down the drain and the sink fills with water. If your roommate left a wad of hair in the drain, the sink fills with water, but more slowly than if you purposefully closed the drain.

Let's say that again. When the drain was open, it had a large lumen. Because it had a large lumen, the resistance to flow was low, and the water went down the pipe. When you plugged the drain, the lumen was zero and the resistance super high, so no water went down the pipe. With the hair clog, there was a smaller lumen than in the open state, but larger than in the plugged, resulting in resistance and flow somewhere between the two extremes. One pipe, one (variable) radius. One arteriole, one (variable) radius.

An arteriole can use its smooth muscle layer to change the caliber of its lumen. It can dilate, making the lumen larger, or it can constrict, making the lumen smaller. Larger lumen, less resistance, more flow. Conversely, smaller lumen, more resistance, less flow. This is how the arteriole controls the flow of blood into the capillary bed it serves.

Resistance, and therefore flow, is controlled by the radii of the arterioles.

Poiseuille's law is an equation you do not need to memorize. But what you should take from it is that the flow through a tube is related to the **radius to the fourth power**. Arterioles are the smallest arteries. They are also the arteries without any other stuff—no adventitia, hardly any elastin or collagen, and only a one-cell-thick endothelium. This allows them to **vary their radius better than any other artery**—from 4 μm to 25 μm. They can change their radius by a factor of 6. Flow changes based on radius size to the fourth power. That means arterioles are capable of varying flow through their lumen by 6^4—a 1,296-fold increase.

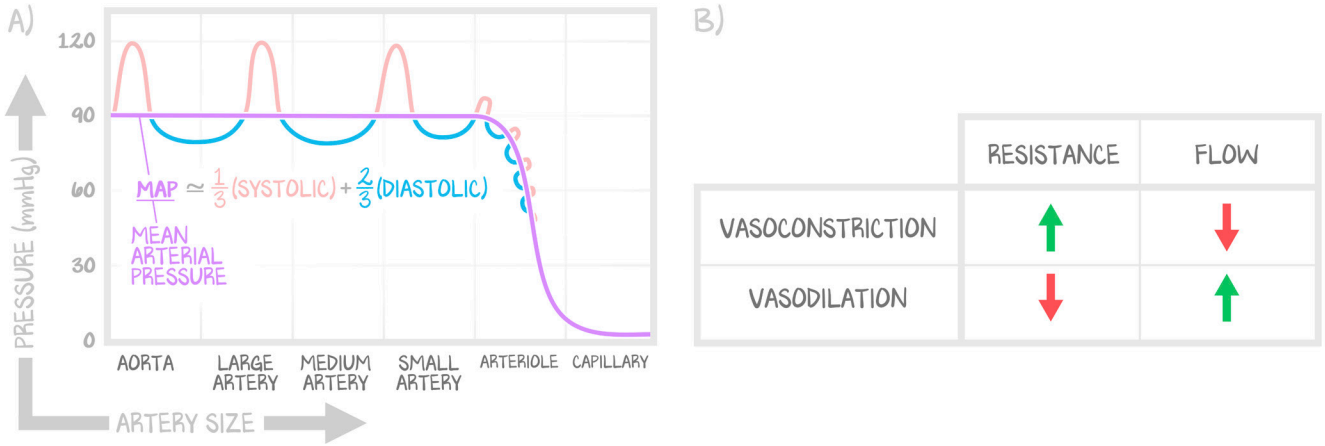

Figure 1.4: Arteries and Arterioles
The purpose of all arteries is to keep the perfusion pressure high enough to get blood to the arterioles, to the tissues. Systole causes peaks in the perfusion pressure, whereas diastole causes the valleys. The MAP is a simpler means of representing blood pressure because it does not vary. An arteriole uses vasoconstriction and vasodilation to control blood flow into the capillary bed that arises from it. If the arteriole dilates, resistance increases and flow decreases. If the arteriole constricts, resistance increases and flow decreases.

This means that, locally, **arterioles control flow to capillaries**. Through chemical signaling, the tissue distal to the arteriole can tell the arteriole whether it needs more or less flow, and the arteriole adjusts. That arteriole can also protect the capillaries if there is a change in pressure—the arteriole changes its smooth-muscle contraction based on the absence or presence of stretch. These mechanisms we cover in detail in Hemodynamics #3: *Blood Pressure Regulation*.

Remember physics class? Resistors, capacitors, and Ohms? How about *in series* and *in parallel*? We're not going to do this like physics class because 98% of students studying to master health and disease didn't like physics class.

This means that, globally, **arterioles control systemic vascular resistance**. *In series* means one after the other. The right ventricle, pulmonary artery, lungs, pulmonary veins, left ventricle are in series. So, too, is the branching arterial tree. The large arteries branch into medium arteries, which branch into small arteries, which branch into arterioles. Those arteries are in series. From the heart to the tissue is in series. *In parallel* means nothing helpful except in physics and circuits, but it means the arterioles can add their cumulative resistance. One small artery doesn't give rise to one arteriole. Arteries continue to branch many times, each time giving off progressively smaller and smaller branches. So one small artery has many arterioles, and that one small artery is just one of many small arteries that branched from the medium-sized artery. Only the arterioles can alter the size of their lumen. All other arteries simply distend and recoil. Every arteriole is *in parallel* with every other because the aorta is the first, largest artery that (eventually) gives rise to all arterioles, so the sum of the resistance in all arterioles is the resistance to blood flow felt by the aorta.

Structure and Function of Arteries

We want you to learn three artery types: large elastic arteries, medium-sized muscular arteries, and microscopic arterioles; each one can be characterized by the relative amount of elastin, collagen, and VSMC in its tunica media, the relative ratio of the size of the lumen to the thickness of the wall, and what's in its adventitia.

Large elastic arteries are the ones that distend and recoil, maintaining the perfusion pressure in diastole. All arteries have the job of bringing the perfusion pressure to the arterioles. Some contribute to that perfusion pressure more than others. We're being rigid with this method. Of course there is a continuum of sizes, from the largest (aorta) to the smallest (arteriole), but we want to remove ambiguity. The large elastic arteries are the ones that contribute the most. Large elastic arteries are the pulmonary artery and the aorta—that's it. In order to be elastic, the **tunica media of the aorta contains mostly elastin**, layered in concentric sheets. To prevent the force of ventricular contraction from blowing the aorta apart, some collagen and some VSMC are present. Elastin allows for recoil, whereas collagen and VSMC allow for high compliance but keep the vessel together. On a cross-section of a large artery, such as the aorta, there is a very large **lumen:media** ratio (12 mm:2 mm in the aorta). These vessels are enormous and require an **adventitia with vasa vasorum**.

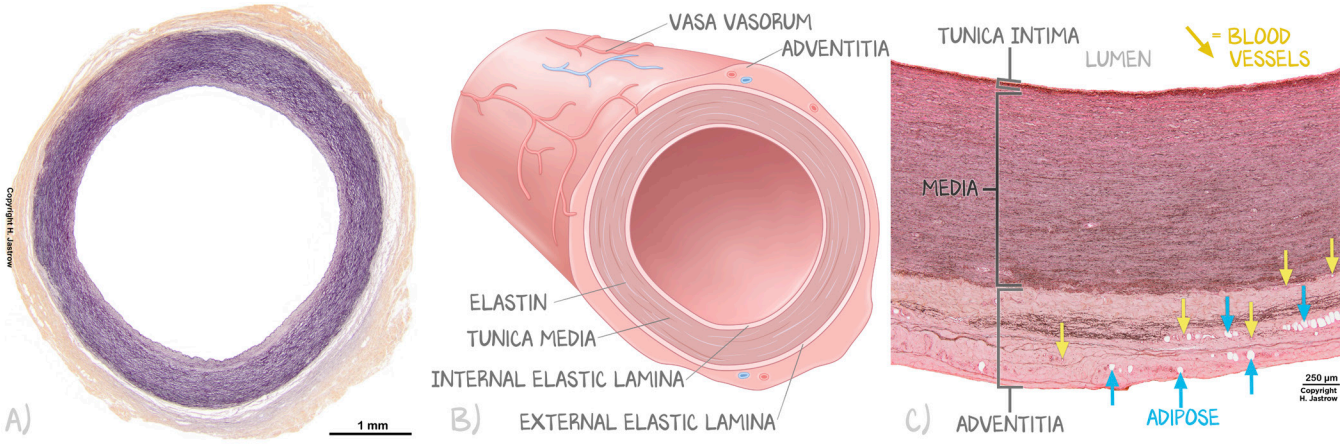

Figure 1.5: Large Arteries
(a) Cross-section of a large muscular artery in its entirety. The tan-colored tissue is the adventitia, representing loose connective tissue, and the purple tissue is the tunica media, with its wavy elastin stained purple. (b) A representative illustration of the aorta as it would be extended from the 2D histology in panel a. The adventitia has blood vessels in it, the tunica media is separated from the intima and adventitia by internal and an external elastic laminae, and the intima is not visible. (c) High-powered view of the aorta showing the lumen (white space at the top), an intima that is barely visible where the lumen ends, a very large media with elastin fibers running left to right, and the adventitia, which has blood vessels and adipose and is made of connective tissue.

Medium muscular arteries. Compared to elastic arteries, muscular arteries present a major shift in histology. The **tunica media** of muscular arteries is almost entirely **vascular smooth muscle cells**. Because the media is smooth muscle cells and very little elastin, the **internal** and **external elastic laminae** are overtly visible when stained for elastin. They need some collagen so that they don't break apart and some elastin to remain flexible, but it is the constant diameter that matters. Just get the blood to the arterioles. The larger ones have a large adventitia. We are being rigid with the method—only large arteries, medium arteries, and arterioles—so we want you to learn that medium arteries have **a smaller adventitia** and a **lumen:media** ratio that is in the middle (2 mm:1 mm). They exist just to get the blood to the tissues they irrigate. The pressures change in the elastic arteries to maintain an overall constant MAP. The muscular arteries obviously do pulsate—you can feel your radial pulse—but they dampen the variation compared to the aorta. This represents **every named artery** other than the aorta and pulmonary artery.

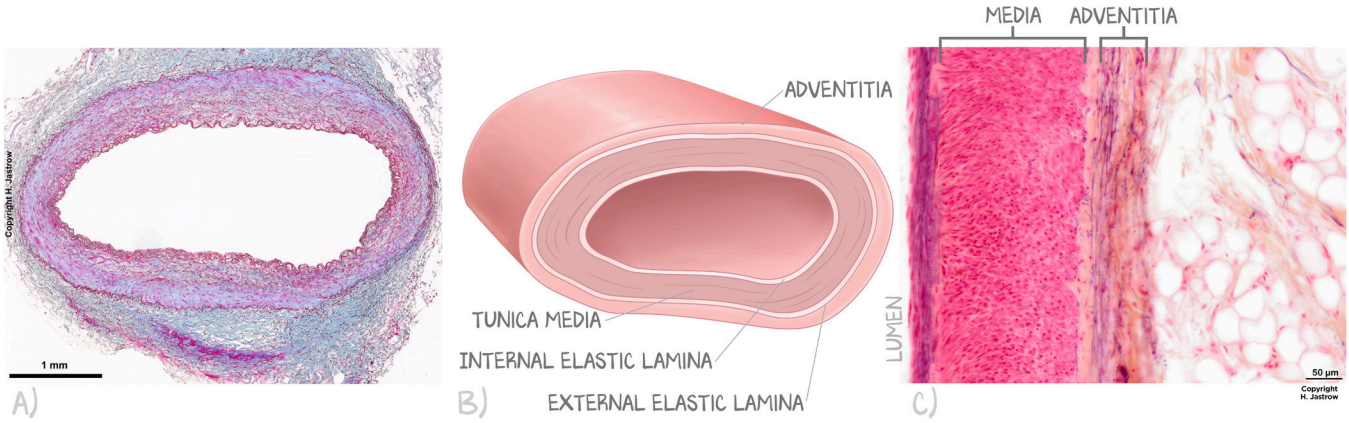

Figure 1.6: Medium Arteries
(a) Low-powered magnification of a medium artery demonstrating a similar arrangement to the large artery—an intima too small to see, a large media, and an adventitia, with the media separated by internal and external elastic laminae. The main difference in appearance is the lumen-to-media ratio. This slide is real histology of a medium muscular artery, although its lumen:media ratio is more akin to that of a large artery. (b) Illustration rendering the 2D histology in panel a as an elongated tube, depicting the blood vessel. (c) High-powered micrograph demonstrating the relative size of the media as compared to the aorta in Figure 1.5.

Arterioles are microscopic—only a few cells thick in total. The endothelial layer is the tunica intima, as with all vessels. The adventitia is absent. They will be found in the connective tissue of other organs on a histological section. Though they are in the connective tissue, the connective tissue is not of the arteriole but of the tissue it connects (between lobules of an exocrine gland, for example). In arterioles, the **tunica media is almost entirely smooth muscle cells**. When the smooth muscle cells contract, the lumen gets smaller. When smooth muscle cells relax, the lumen gets bigger. Because arterioles can vary their diameter so much, the lumen:media ratio isn't static. But the **media is usually larger** than the lumen.

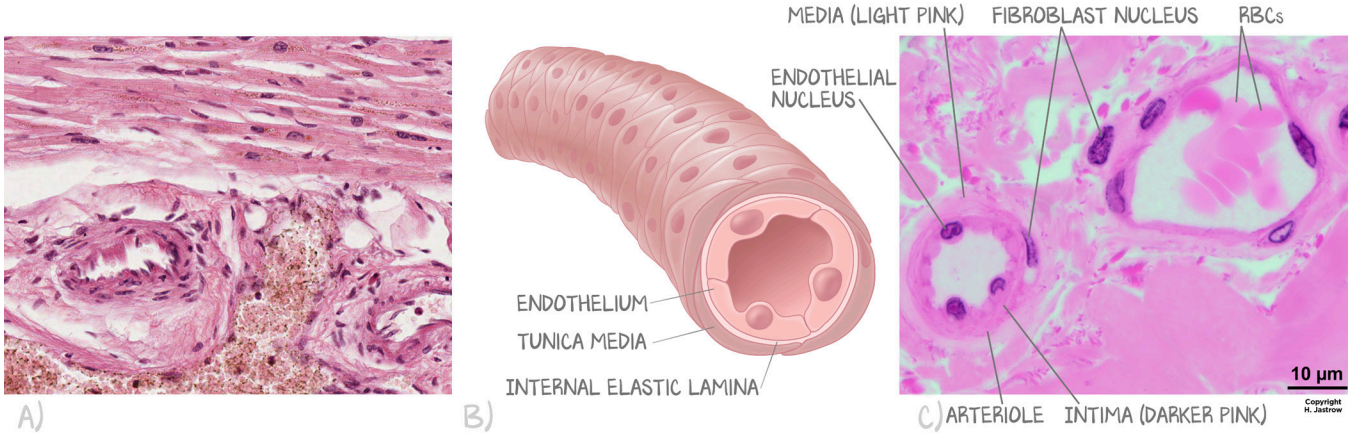

Figure 1.7: Arterioles
(a) The lowest magnification at which arterioles can be seen. There is a thick media with a small lumen. (b) Illustrated tubular arteriole with endothelium and a very thin layer of VSMC represents the 2D histological arteriole on the left side of panel c. (c) High-powered histology demonstrating an arteriole on the left (circular shape) and a nearby venule (larger lumen, floppier, with RBCs in it).

The arteries we don't talk about. As the arterial tree gets farther from the heart, there is a gradual progression in the necessity of function, and therefore a gradual progression in the structure of the tunica adventitia and tunica media. Know that there is a gradual progression, but learn that there are three completely distinct artery types. We aren't going to mention the in-between artery types. We aren't

going to talk about small arteries. A larger small artery looks like a medium-sized artery. A smaller small artery looks like an arteriole. When there is overlap in structure and function, it is impossible to test you as to a "single best answer," and it doesn't help understand how flow and pressure regulation influence perfusion in the body. We have removed ambiguity and suggest that is how you should learn it.

Structure and Function of Veins

Veins carry blood to the heart from the periphery. The venous system is an **extremely low-pressure system** and has **no forward-driving force** to propel blood coming from the arterial system behind it. The large arteries store and recoil driving force, the medium arteries are conduits, and the arterioles crank up the resistance so that the pressure in the capillary bed is where it needs to be. The hydrostatic force is used up within the capillary. There cannot be any force left in the veins. In addition, for the parts of the body below the heart (which is most of the body), veins must contend with gravity, a pulling force that causes blood to pool in the veins. Yet blood does make it back to the heart. We'll explore how next.

There are three main types of veins: venules that receive blood from capillaries, medium veins, and large veins.

Veins, in general, are **capacitance vessels**. That means they are loose and floppy. They are made to be filled. They do not need to resist the pressures of the ventricles, so they do not need to distend and snap back with elastic recoil. Therefore, they need **very little elastin** (they do not need to distend) and **very little smooth muscle** (they do not dilate or constrict to regulate flow). Therefore, the tunicae (intima, media, and adventitia), which are clearly demarcated in the arteries, are less prominent and harder to distinguish in veins. Veins often run alongside their arterial counterpart in the same connective tissue. Arteries are distended, appearing circular on cross-section. Veins, on the other hand, tend to be floppy and appear unfilled. The presence of the artery helps to identify the vein near it.

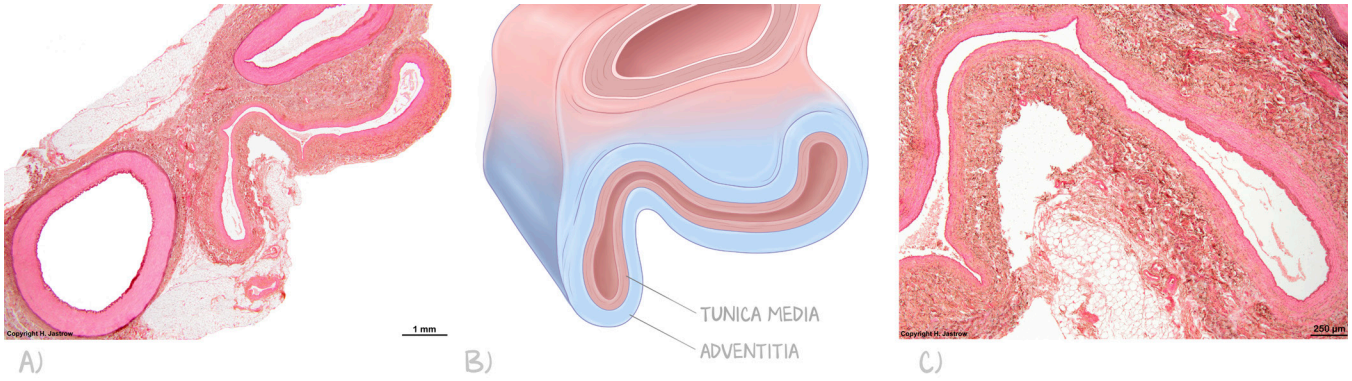

Figure 1.8: Veins
(a) At low magnification, two large arteries (one at the bottom left and one cut off on the top right) maintain a generally circular shape. Nearby is the non-distended, flattened lumen of a floppy vein. (b) The illustration mirrors the relationship seen in panel a. The artery with a large lumen and thick tunica media compared to those of the collapsed and thin-walled vein. The intervening connective tissue (the pink around the artery and the blue around the vein) nebulously mixes between the blood vessels. (c) High-magnification view of the collapsed vein; the lumen is white, the tunica media is pink, and the surrounding connective tissue is brown.

Venules. Venules are the venous equivalent of arterioles. They are small, having only a few layers of smooth muscle in their tunica media. Just like vasoconstriction, venoconstriction is controlled by the autonomic nervous system, and a change in venules' diameter can squeeze blood along the venous system—there is a small driving force pushing blood through the veins. There are specialized **high endothelial venules** that are post-capillary venules without any smooth muscle layer, which permit lymphocyte migration out of capillaries and into secondary lymphoid organs.

Medium veins. Medium veins are named for the artery they run along. Medium veins have **valves**, especially in the lower extremities. Valves are one-way doors. Any blood pushed toward the heart needs to be kept heading in that direction. **Skeletal muscle contraction** generates the driving force upward through the veins. Valves exist to prevent the backflow of blood, to resist the pull of gravity. The three tunicae are most evident in the medium veins, which do have smooth muscle layers, though they are still capacitance vessels and appear floppy on histology.

Large veins. Large veins have a **massive adventitial layer**. They are capacitance vessels as well. But should blood back up into large veins, as in heart failure, for example, these vessels will fill. They do not distend and recoil; they fill. As these large vessels fill, they will appear engorged—visible but not pulsating. They resist distension because of the large amount of **collagen** in their **tunica media**. You can think of arteries as elastic bands, stretching and snapping back, and veins as garden hoses. The first time you turn the water on, you see the garden hose shake a little as the water runs through it. When you turn the spigot on, water has to run through the hose before it comes out the other side. If you block the end of the hose with your hand, and the water is on, the hose does not expand the way a water balloon would. You might see the pressure cause the water to leak out of a hole in the hose, or backpressure force water out a poor connection at the spigot, but the hose does not change shape. Veins are the hose. They can be filled. When they are filled, they won't stretch. If water keeps coming from the house, the entire hose will fill. So too do the veins. They are capacitance vessels because they start unfilled and floppy. They are not distensible because they do not stretch when filled.

Side note: in right-sided heart failure, blood backs up in the superior vena cava. This is translated to the internal jugular vein. Because it is so near the heart, there will be visible jugular vein pulsations. This is not a product of ventricular force but of the opening and closing of the tricuspid valve—when the vein drains a little during diastole and then re-engorges during diastole, there is the appearance of venous pulsations. It is not the distension and recoil of arteries, but there are visible waves that got named pulsations. We'll cover this more in heart failure.

Flow through Arteries: Turbulent vs. Laminar Flow

This section is obligatory. The science behind it can be a little esoteric, so it is left out of the video. But its consequences—valve murmur and arterial bruit (*"broo-EE"*)—are not esoteric or trivial.

Blood flows down the arterial tree. Arteries bring blood from the heart to the tissues. But HOW does blood move? Systolic contraction delivers longitudinal force during systole, forcing blood forward. In diastole, the elastic arteries recoil and translate their transmural force to longitudinal force. When there are no obstructions to flow, when blood is flowing in the tubes of the artery, flow is **laminar**. Laminar flow happens when there is a steady rate of flow through long, smooth blood vessels. Laminar blood flows in **streamlines**, with each layer of blood remaining the same distance from the vessel wall. An RBC at the top of a blood vessel doesn't dart down to the bottom in a streamline; that RBC stays at the top of the blood vessel. When laminar flow occurs, the velocity of flow in the center of the vessel is far greater than it is along the outer edges. This phenomenon is called **parabolic velocity**. The molecules in contact with the vessel wall flow slowly because they adhere to the vessel wall. The next layer slips over those molecules, faster than the first layer. The next layer slips still faster. This is why inflammation leads to venodilation, stagnation of blood flow, to allow the leukocytes in the center of the lumen to marginate to the edges of the vessel wall (Immunology #4: *Innate Immune Response*). The dilation of arteries ensures that streamlines rapidly deliver blood to the tissues. The dilation of veins causes stagnation at the point of inflammation to allow the cells to get out of their streamline to reach the endothelium.

Turbulent flow is the opposite of laminar flow. Turbulent flow occurs either when there is either too much flow through the same diameter or normal flow through a narrowed vessel lumen. We can use this to detect blood pressure—the cuff compressing the artery so that the turbulent flow can be heard, the Korotkoff sounds. Turbulent flow also occurs in disease—atherosclerotic narrowing of a vessel presenting as a bruit.

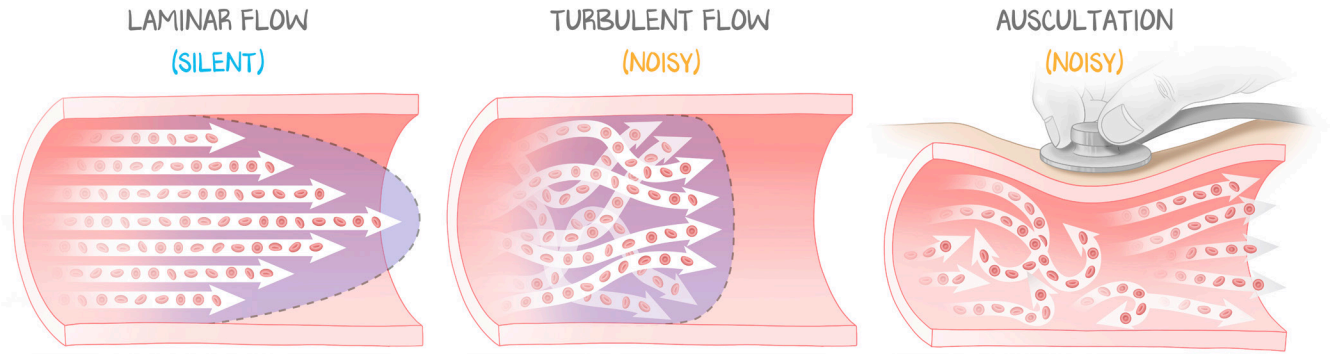

Figure 1.9: Laminar and Turbulent Flow
Laminar flow propels blood in streamlines, the fastest cells traveling in the center streamline. Laminar flow is silent. Turbulent flow (in this illustration not caused by any pathology, merely depicting nonlinear streamlines) is the flow when blood is not in clear streamlines. Blood still flows, but it makes noise. If there is turbulent flow from a pathology, there will be a bruit when a stethoscope is applied over the lesion. You can also do this to yourself, auscultating the femoral artery, then depressing the stethoscope firmly down on the artery, temporarily partially occluding the artery. When you let go, the sound will go away.

There are many complicated uses of turbulent and laminar flow. Blood pressure is pulsatile. Physics in real blood vessels is not as simple as "control resistance, control flow." When you get to the extreme invasive measuring of a patient's hemodynamics—an action that 95% of clinicians will never even see—the equipment relies on an accurate understanding of blood flow. Measuring a patient's systemic vascular resistance and central venous pressure and calculating their cardiac output with the Fick equation are necessary for the patient with end-stage heart failure who just had an LVAD placed yesterday and is post-op waiting for their transplant on two vasopressors. **This is not for you.** And actually, the machine does it for you, reporting a cardiac output.

This simplified version of turbulent and laminar flow is used to show you that **your ears can detect disease** (bruits, murmurs) when flow becomes turbulent, and that turbulent flow damages the endothelium, predisposing to acute thrombosis, the pathogenesis of acute coronary syndrome (a heart attack).